

Chapter 29

PELVIC PAIN

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INTRODUCTION AND TAXONOMY

(Box 29–1)

Chronic pelvic pain (CPP) of nonmalignant origin may be defined as nonmenstrual pelvic pain of 6 months or more duration that is severe enough to cause functional disability or require medical or surgical treatment. Acute pelvic pain and CPP may be localized or referred and can be present in all age groups. Further, CPP may be divided into syndromes of the pelvic cavity (diffuse pain with no particular pelvic structure identified or hypothesized organ involvement such as irritable bowel syndrome/interstitial cystitis) or pelvic floor (prostatodynia, orchialgia, vulvodynia, urethral syndrome). Pelvic pain is one of the most challenging problems faced by health care practitioners because it is difficult to identify both an etiology and, subsequently, a treatment. It is often associated with other problems such as voiding difficulty or sexual dysfunction. Before intervening, a comprehensive evaluation and work-up are necessary. Practitioners involved in the treatment of pelvic pain include family physicians, internists, gynecologists, gastroenterologists, psychiatrists, neurologists, urologists, and pain medicine specialists.

The pelvis contains several anatomic structures including muscles that form the pelvic diaphragm and the urogenital diaphragm, the urinary bladder, distal ureters, sigmoid colon, rectum, some small intestine, internal genitalia, blood vessels, lymph vessels, lymph nodes, and nerves.

EPIDEMIOLOGY

Pelvic pain is a common disorder in women, especially in the reproductive age group, with an estimated prevalence of 3.8% in women aged 15 to 73, resulting in about 10% of all referrals to gynecologists. This prevalence rate parallels those rates reported for asthma, low back pain, and migraine headaches. Approximately 15% to 20% of women aged 18 to 50 years have CPP of greater than 1 year's duration. In fact, the lifetime occurrence rate of CPP may be as high as 33%. Patients with CPP may exhibit pathology that fails to correlate with the pain, and one third to one half of cases may exist with no identifiable pathology. Signs and symptoms of CPP may be evident in at least 50% of patients with preexisting sexual or physical abuse. The psychological literature suggests that women with CPP report greater psychological distress.¹ For instance, women with CPP have elevated rates of depression, anxiety, hostility, and somatic complaints. The direct health care-related costs owing to CPP are estimated to be \$880 million per year.² Both direct and indirect costs potentially amount to greater than \$2 billion per year. The individual burden of CPP ranges from years of personal suffering to missed or lost work, marital or relationship disruption, and multiple medical and/or surgical interventions.

PELVIC NEUROANATOMY AND PATHOPHYSIOLOGY

Many potential areas of pathologic involvement exist in patients with CPP, and despite extensive diagnostic investigation, the symptoms often remain puzzling. The pelvis is a complex, neurophysiologic region, and sources of pain may arise from somatic, visceral, and/or neurogenic structures. Somatic sources include muscles, pelvic bones and joints, ligaments, and fascia. Visceral sources of CPP include the reproductive, gastrointestinal, and urologic systems (Fig. 29–1). Broadly, dual projections from the thoracolumbar and sacral portions of the spinal cord innervate the pelvis and then coalesce into neuronal plexuses that send fibers throughout the pelvis. The pelvic viscera receive neurons from the sympathetic (thoracolumbar) and parasympathetic (craniosacral) systems. More specifically, visceral afferent fibers that travel in the sympathetic trunk contain their cell bodies in the dorsal root ganglia between T10 and L2. Similarly, visceral afferents traveling with parasympathetic fibers contain their cell bodies in the dorsal root ganglia between S2 and S4. The lateral pelvic region transmits pain through parasympathetic neurons arising from S2–S4, whereas the midline structures of the pelvis receive most of their input from sympathetic fibers arising from T10–L1. These fibers coalesce to form the superior hypogastric plexus (SHP or presacral nerve), which lies immediately anterior to sacral promontory at the L5–S1 level. The SHP transmits sensory input from the descending and sigmoid colon, rectum, vaginal fundus, bladder, prostate, prostatic urethra, testes, seminal vesicles, uterus, and ovaries. This plexus then divides into hypogastric nerves that eventually form the inferior hypogastric plexus (IHP). The IHP is the primary neural, autonomic coordinating center in the pelvis that integrates both parasympathetic and sympathetic output. Efferent fibers that derive from the IHP innervate the clitoris, vagina, and urethra in women and the prostate, seminal vesicles, vas deferens, epididymus, and penis in men.

The termination of the paired paravertebral sympathetic chains occurs at the sacrococcygeal junction and is called the *ganglion impar* (ganglion of Walther). The neuronal interconnections of this ganglion are poorly understood, although blocking this ganglion can provide relief of pain originating from the perineum or distal pelvic structures. Other regions believed to be subserved by the ganglion impar include the distal rectum and anus, distal urethra, vulva, and distal third of the vagina. Note that the sympathetic nervous system is contiguous; therefore, there may be overlap and variation of innervation among ganglia (e.g., lumbar ganglia, hypogastric plexus, ganglion impar).

Painful sensations from the pelvis to the brain travel through dorsal root ganglion cells (sensory neurons) located in the thoracolumbar and sacral portions of the spinal cord. Animal studies suggest that pelvic sensations are carried within the sacral parasympathetic system to a greater extent than the thoracolumbar sympathetic system.³ Certain CPP syndromes such as interstitial cystitis, irritable bowel syndrome, and prostatodynia (prostatitis) may be associated with neurogenic inflammation. This process is initiated by neuropeptides such as substance P, calcitonin gene-related peptide (CGRP), and neurokinin A and B, which are released by primary afferent neurons. Primarily C-fibers and some Aδ-fibers are believed to be the primary afferent fibers involved in the process of neurogenic inflammation. Within the pelvis, neurogenic inflammation has been described in the digestive, genitourinary, and reproductive systems. Some researchers have hypothesized that referred pain associated with interstitial cystitis and vulvodynia or interstitial

Box 29–I CURRENT DIAGNOSIS

1. Pelvic pain has been defined as nonmenstrual pelvic pain of 6 months or more that is severe enough to cause functional disability or require medical or surgical treatment.
2. Before intervening, a comprehensive clinical evaluation is necessary.
3. Practitioners involved in the treatment of pelvic pain include family physicians, internists, gynecologists, urologists, psychiatrists, and pain medicine physicians. Chronic pelvic pain (CPP) is often associated with voiding difficulty or sexual dysfunction.
4. Many areas of CPP pathology exist, and the etiology is frequently puzzling. In one third to one half of cases, pelvic pain persists without evident pathology. Even when pathology exists, it may not correlate with painful symptomatology.
5. Imaging is often nonrevealing because many CPP conditions fail to present on film.
6. Over 40% of diagnostic laparoscopies are performed for CPP and may reflect a reasonable approach to treating refractory pelvic pain.
7. Specific nerve blocks in the pelvis such as superior hypogastric plexus (SHP) and ganglion impar may aid in identifying the region of pain.

cystitis and prostatodynia may represent certain mechanisms of neurogenic inflammation when patients report referred pain in the urogenital floor (referred area) of the bladder.⁴ Further, animal studies suggest that a neurogenic inflammatory mechanism may explain edema and trophic alterations in the skin, muscle, and subcutaneous tissue of patients reporting discomfort in referred zones from chronic visceral pain.

Psychological disorders such as somatization, psychosexual dysfunction, and depression can contribute to CPP and merit a comprehensive evaluation by trained mental health professionals. More often, diseases of several systems in the body may account for symptoms of CPP, and sometimes, disorders of more than one system may contribute to ongoing pelvic discomfort (Table 29–1). These disorders can cause a constellation of symptoms ranging in severity from producing no pain to causing debilitating, persistent pain. Such disparate responses may result from changes in visceral nerve function or from altered central nociceptive processing; however, precise mechanisms remain unclear.

CLINICAL FEATURES

A careful and detailed history and physical examination are essential in the diagnosis and treatment of CPP. Demographic profiles suggest that women with CPP are no different from women without pelvic pain in terms of age, race, ethnicity, education, socioeconomic status, or employment status. Certain factors, however, can increase the risk of developing pelvic pain, which a thorough history can elicit. For example, in addition to assessing typical pain descriptors such as location, severity, quality, and timing, clinicians should include an obstetric history that details the patient's menstrual cycle, dysmenorrhea, dyspareunia, and any voiding dysfunction. Moreover, pregnancy and childbirth are specific events from which CPP may arise. In particular, patients with lumbar lordosis, delivery of a large infant, pelvic muscle weakness, poor physical conditioning, and a difficult delivery represent a group at higher risk of developing CPP. In nulliparous patients, several conditions may predispose them to developing CPP. For instance, diseases such as endometriosis, chronic pelvic inflammatory disease, interstitial cystitis, and irritable bowel syndrome may lead to complaints of CPP. Although a detailed psychosocial evaluation is best performed by a psychiatrist or psychologist, the pain specialist can elicit a brief psychological history. Focusing on depressive symptomatology is important and constitutes one of several predictors of pain severity in women suffering from CPP. Coexisting depression also serves as an indicator of response to treatment; that is, patients

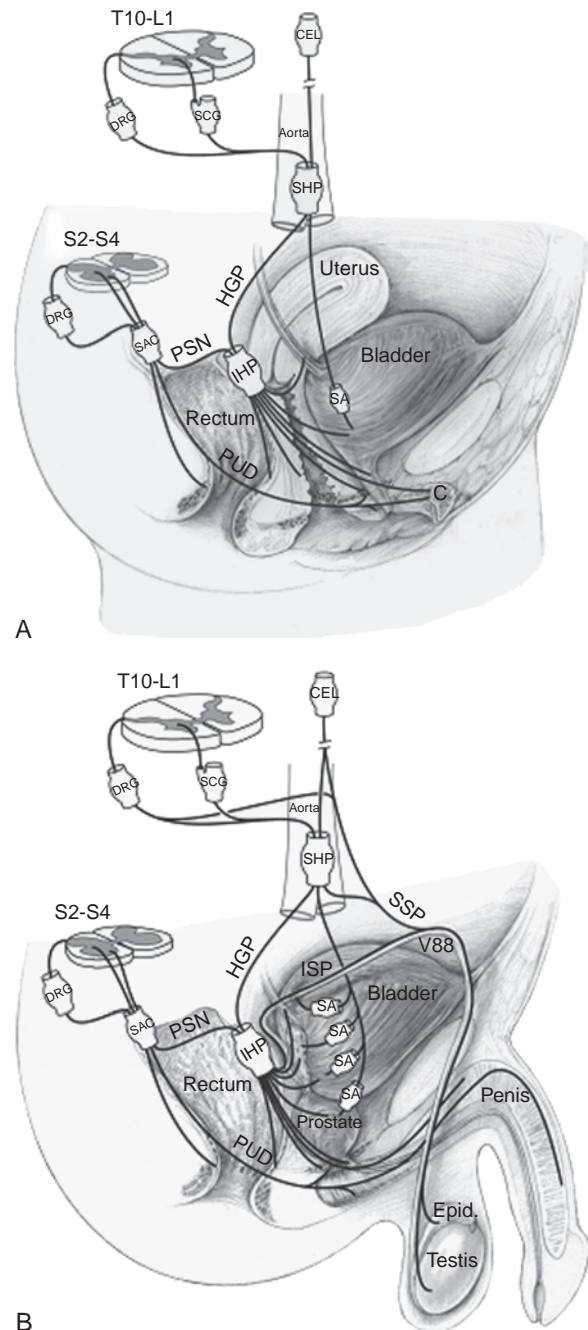


Figure 29–I. Pelvic neuroanatomy of females (A) and males (B). Animal data have provided much of the anatomic information about human pelvic innervation. CEL, celiac plexus; DRG, dorsal root ganglion; HGP, hypogastric plexus; IHP, inferior hypogastric plexus; ISP, inferior spermatic plexus; PSN, pelvic splanchnic nerve; PUD, pudendal nerve; Epid., epididymis; SA, short adrenergic projections; SAC, sacral plexus; SCG, sympathetic chain ganglion; SHP, superior hypogastric plexus; SSP, superior spermatic plexus. (From Wessellmann U, Burnett A, Heinberg L, et al. The urogenital and rectal pain syndromes. *Pain* 1997;73:269–294. Used with permission.)

with depression tend to benefit less from treatment strategies than those without depression. Finally, a comorbid history of abuse is important to uncover owing to the association between physical and sexual abuse and the subsequent development of CPP. The International Pelvic Pain Society has designed a comprehensive

Table 29–I. Systems Associated with Potential Sources of Pelvic Pain

Gynecologic	Urologic	Gastrointestinal	Musculoskeletal	Neurologic
Endometriosis	Interstitial cystitis	Irritable bowel syndrome	Pelvic floor dysfunction	Postherpetic neuralgia
Adenomyosis	Malignancy	Inflammatory bowel disease	Myofascial pain syndromes	Incisional neuroma
Pelvic congestion syndrome	Radiation cystitis	Chronic constipation	Adhesions	Visceral hyperalgesia
Adhesions	Acute recurrent cystitis	Colon malignancy	Fibromyalgia	Pudendal neuralgia
Ovarian remnant syndrome	Chronic urinary tract infection	Colitis	Muscular sprains (rectus muscle)	Entrapment neuropathy
Pelvic inflammatory disease	Urolithiasis	Hernia	Piriformis syndrome	
Malignancy	Uninhibited bladder contractions	Diverticular disease	Spondylosis	
Leiomyomata	Urethral syndrome	Diarrhea	Chronic coccygeal pain or back pain	
Symptomatic pelvic relaxation	Urethral caruncle			
Cervical stenosis	Urethral diverticulum			
Adnexal cyst				
Postoperative peritoneal cyst				
Tuberculous salpingitis				
Gärtner's cyst				
Benign cystic mesothelioma				

history and physical examination document that targets CPP patients.⁵ The website may be accessed at www.pelvicpain.org.

Physicians may view the clinical presentation of patients with CPP as confusing and often enigmatic. CPP patients (women and men) often struggle in describing and localizing their pelvic pain, and clinicians feel limited in examining and quantifying CPP. Furthermore, the differential diagnosis of CPP is vast because patients report symptoms that appear diffuse and nonspecific. In part, CPP reflects a visceral pain condition, and patients generally describe visceral pain as “deep,” “dull,” “squeezing,” “crampy,” and “oppressive.” In contrast to somatic pain, mechanical stimulation, ischemia, and chemical irritation may induce visceral pain.

Visceral pelvic pain displays three characteristics: (1) true visceral pain: experienced deep in the pelvis; (2) referred visceral pain: sensed in somatic regions such as muscle and skin; and (3) referral site hyperalgesia: the site of referred pain may demonstrate increased sensitivity to a painful stimulus. Some have suggested that both visceral and somatic primary afferent neurons may converge on similar neurons in the spinal cord and thereby cause the phenomenon of referred pain.⁶ This overlapping segmental afferent innervation may explain why patients report referred pelvic pain to the perineum, abdomen, thighs, legs, and back. In fact, the process of referred pain may manifest itself as a sensation that several organs of the pelvis feel affected during the course of CPP states.

EVALUATION

The physical examination should be thorough and evaluate the musculoskeletal, gastrointestinal, and urinary systems in addition to the reproductive tract. Ideally, the examination should be performed while the patient is standing, sitting, in the prone position, and in the lithotomy position to fully evaluate all of these systems.

The physician should focus on locating foci of tenderness and correlating them with areas of pain.

There are no standard diagnostic tests that should be ordered on all patients with CPP. Rather, the history and physical examination should direct relevant testing. There is an array of imaging and blood testing that clinicians may order, and a partial list of tests that correlate with suspected diagnosis and symptomatology is presented in Table 29–2. Laparoscopy may be of value as well. Actually, over 40% of diagnostic laparoscopies are performed to help clarify the etiology of CPP. Endometriosis and adhesions encompass the vast majority of laparoscopic diagnoses, whereas therapeutic removal of endometrial lesions or lysis of adhesions can be performed during laparoscopic surgery. A newer, state-of-the-art diagnostic laparoscopic technique has emerged in which the patient is awake for the procedure and can provide feedback about pain intensity while the surgeon gently probes specific pelvic regions.² The value of this test has not been demonstrated, however.

MANAGEMENT

Although a single pathologic process may be responsible for a specific CPP syndrome and can be treated, most CPP is caused by a number of factors for which multiple treatments are needed. The mainstays of treatment include noninvasive, pharmacologic, and surgical interventions (Box 29–2).

Complementary/Alternative Medicine

Noninvasive treatments consist of exercise/physical therapy, cognitive-behavioral techniques, massage, dietary modification, and acupuncture. These modalities may be beneficial in many chronic pain

Table 29–2. Imaging or Laboratory Testing That May Be Appropriate to Support a Specific Diagnosis of Chronic Pelvic Pain

Suspected Diagnosis/Symptom	Tests
Adenomyosis	US, hysterosalpingography, MRI
Chronic urethral syndrome	Urodynamic testing
Entrapment neuropathy	EMG/nerve conduction studies
Constipation	Thyroid function tests, renal function tests, hepatic function tests, electrolytes
Diarrhea	Test for ova and parasites in stool, stool guaiac, stool culture, barium enema, upper GI series, CT
Diverticular disease	Barium enema
Dyspareunia	Gonorrhea and chlamydia cultures, vaginal and urine cultures, vaginal wet preparation, vaginal pH
Endometriosis	CA 125, US, barium enema, CT, MRI, laparoscopy
Hernia	Abdominal wall US, CT, herniography
Interstitial cystitis	Cystourethrography, KCl bladder challenge test, urine cultures, urine cytology, urodynamic testing, bladder biopsy
Ovarian remnant syndrome	FSH, estradiol, GnRH stimulation test, US, barium enema, CT
Ovarian retention syndrome	US, CT
Pelvic congestion syndrome	Pelvic venography, US, MRI
Tuberculous salpingitis	Chest x-ray, PPD
Urethral diverticulum	US, voiding cystourethrography, MRI
Gärtner's cyst	US, MRI

CA, cancer; CT, computed tomography; EMG, electromyography; FSH, follicle-stimulating hormone; GI, gastrointestinal; GnRH, gonadotropin-releasing hormone; MRI, magnetic resonance imaging; PPD, purified protein derivative; US, ultrasound.

conditions. Exercise, physical therapy, and massage can ease the severity of CPP, given that many CPP patients display coexisting myofascial dysfunction and may even develop kinesophobia. Dietary modifications may help control certain painful symptoms if they are associated with conditions such as irritable bowel syndrome or interstitial cystitis. Cognitive-behavioral techniques may not alter the disease process, but they certainly help reframe the role CPP has taken in an individual's life and move patients toward healthy, functional living.

Pharmacologic Treatment

Evidence supports the use of nonopioid analgesics such as nonsteroidal anti-inflammatory drugs (NSAIDs) and acetaminophen in treating a variety of pain conditions. Many agents are recommended for initial treatment in cancer pain by the World Health Organization, and we can apply their benefit in this broad group of cancer patients to the nonmalignant pain population. Moreover, certain neuropathic agents such as gabapentin (Neurontin) and the tricyclic antidepressants (TCAs) have demonstrated value in treating neuropathic pain states (postherpetic neuralgia, diabetic painful neuropathy) and, by extension, may be helpful in controlling CPP. There are no clinical trials addressing the efficacy of NSAIDs in CPP, although clinicians may consider them as reasonable first-line medications given their proven, moderate analgesic efficacy in cancer pain conditions.

TCAs and other antidepressants are frequently used in the treatment of neuropathic pain and other chronic pain states. Clinicians should consider the TCAs as useful agents for patients suffering from CPP. TCAs (amitriptyline, nortriptyline, desipramine, imipramine, and doxepin.) in particular may be effective. However, only one study has documented the effectiveness of TCAs in CPP. Walker and coworkers⁷ studied 14 women with CPP and treated them with increasing doses of TCAs to a maximum dose of 100 mg per day. Seven of the women dropped out owing to adverse effects, although 6 of the remaining 7 remained pain free at 1 year. Another

small study by Engel and associates⁸ examined sertraline (Zoloft), a selective serotonin reuptake inhibitor (SSRI) for the treatment of CPP. This was a double-blinded, placebo-controlled, cross-over study using 50 mg of sertraline once a day. The researchers found no difference in pain scores between the two groups. The SSRI group of antidepressants appears to possess the least analgesic properties versus TCAs and selective serotonin and norepinephrine reuptake inhibitors (SNRIs). Despite the lack of convincing evidence

Box 29–2 CURRENT THERAPY

Therapy is achieved through a multimodal approach with a goal of functional restoration. The mainstays of treatment include noninvasive, pharmacologic, surgical, and nonsurgical interventions.

1. Noninvasive treatments consist of exercise/physical therapy, cognitive-behavioral techniques, massage, dietary modification, and acupuncture.
2. Pharmacologic therapies for CPP include these types of medications:
 - a. Nonsteroidal anti-inflammatory drugs (NSAIDs; e.g., acetaminophen, ibuprofen, celecoxib).
 - b. Tricyclic antidepressants (TCAs; e.g., amitriptyline, nortriptyline, desipramine).
 - c. Anticonvulsants (e.g., gabapentin, pregabalin, topiramate)
 - d. Opioids (e.g., methadone, oxycodone, morphine, transdermal fentanyl, oxycodone); focus on long-acting opioids ± short-acting agents for breakthrough pain.
 - e. Hormone agonists (gonadotropin-releasing hormone [GnRH] agonists, progestins).
3. Surgical interventions
 - a. Evidence suggests that laparoscopic excision or destruction of endometriotic tissue reduces pain compared with diagnostic laparoscopy alone.
 - b. Denervation procedures: Presacral neurectomy: excision of the superior hypogastric plexus (SHP; presacral nerve), or laparoscopic uterosacral nerve ablation (LUNA)
4. Nonsurgical options
 - a. SHP blocks or neurolysis (chemical or thermal).
 - b. Ganglion impar blocks or neurolysis (chemical or thermal).

for the use of antidepressants in CPP, these remain a rational and safe option for CPP sufferers.

Anticonvulsant medications have shown efficacy in treating neuropathic pain such as diabetic neuropathy and postherpetic neuralgia. Their pain-relieving properties are applied to other neuropathic pain states including CPP. Practitioners may consider several anticonvulsants such as gabapentin, pregabalin (Lyrica), lamotrigine (Lamictal), topiramate (Topomax), and tiagabine (Gabitril). Gabapentin may be particularly useful in treating CPP because researchers have demonstrated its effectiveness in controlling pain related to the genitourinary tract.⁹

Opioids for the treatment of nonmalignant chronic pain remain controversial. Opioid maintenance therapy should be considered after failure of other reasonable therapies and if pain is impeding all efforts for improved function. Some clinical trials suggest a good analgesic response to opioids in chronic pain, but not necessarily a functional or psychological improvement.¹⁰ In contrast, clinical experience in pain centers suggests that opioid therapy may improve function without significant adverse effects when physicians have exhausted other treatment modalities.¹¹ Prior to initiating opioids, clinicians should strongly consider implementing an informed consent for opioid therapy that details risks and benefits of treatment, mandates only one prescriber, and highlights the process as a trial of opioid therapy that can be discontinued at the physician's or patient's discretion. Close and regular follow-up to assess treatment effectiveness in terms of pain relief, improved function, enhanced quality of life, and adverse effects along with proper urine drug monitoring is essential to responsible opioid therapy.

Hormone therapy in the form of oral contraceptives, gonadotropin-releasing hormone (GnRH) agonists, and progestins has been incorporated with some success in select CPP states. For instance, oral contraceptives are recommended for pelvic pain associated with endometriosis by the American College of Obstetricians and Gynecologists, even though supportive evidence is lacking. Oral contraceptives suppress ovulation, markedly reduce uterine activity, and reduce the pain associated with menses; hence, they may reduce pain related to other gynecologic conditions. GnRH agonists (nafarelin, goserelin, and leuprolide) down-regulate hypothalamic-pituitary gland production resulting in decreased estradiol levels. Numerous trials demonstrate that these medications are effective in treating pelvic pain caused by endometriosis,¹² and observational data suggest that GnRH agonists are effective in treating pelvic pain resulting from ovarian retention syndrome and ovarian remnant syndrome. Principal adverse effects include bone density loss, which can be reversed with progesterone replacement with or without estrogen.^{13,14} Finally, progestins (medroxyprogesterone acetate) are effective in the treatment of pain associated with endometriosis as well as pelvic congestion syndrome.

The previously mentioned medications may have deleterious effects; therefore, clinicians must carefully select patients before beginning any of these medications.

Surgical Interventions

Surgical options either treat the primary problem or strive to alleviate the pain, although the latter is usually of unknown origin. Surgery typically treats pain resulting from endometriosis or adhesions. Evidence suggests that laparoscopic excision or destruction of endometriotic tissue reduces pain compared with diagnostic laparoscopy alone.¹⁵ Unfortunately, recurrence rates approach 100%, with an average time to recurrence of 40 to 50 months.¹⁵ Recurrence may be reduced with thorough excision of endometrial tissue and proper medical management. Because adhesions are commonly believed to cause CPP, adhesiolysis remains an option for patients with known suspected adhesions. One observational study suggested that up to 85% of women report reduced pain

after adhesiolysis.¹⁶ Conversely, a randomized trial of 48 women with laproscopically diagnosed adhesions receiving either conservative management or laproscopic adhesiolysis showed no significant difference in pain relief at 1 year.¹⁷ Thus, the efficacy of this procedure remains unclear. Hysterectomy remains an option for women suffering from intractable CPP. Approximately 10% to 12% of hysterectomies are performed for CPP. Results of the Maryland Women's Health study and the U.S. Collaborative Review of Sterilization suggest that 75% to 90% of women report pain relief for greater than a year after hysterectomy.¹⁸ These data indicate that woman with CPP of gynecologic origin may respond well to hysterectomy.

For patients without a definable disease, surgical denervation may offer pain reduction. This procedure consists of surgical transection or excision of peripheral nerves. Lesioning of the central nervous system has been performed, but in only extreme cases of CPP and without convincing data to support its routine use. A more common procedure, presacral neurectomy, involves excising the SHP (presacral nerve). Surgeons incise the pelvic peritoneum over the sacrum and identify and transect the SHP. The SHP supplies the cervix, uterus, and proximal fallopian tubes; consequently, SHP neurectomy may be more useful for patients affected with severe dysmenorrhea or endometriosis. Nonetheless, this surgical approach has been used to treat CPP of unknown origin. Clinical experience suggests a better response in patients with midline pelvic pain than with lateral pelvic pain. In a retrospective review examining 655 patients undergoing presacral neurectomy for dysmenorrhea or chronic pain of unknown origin, 72% of the 392 patients with a diagnosis of dysmenorrhea reported significantly decreased pain.¹⁹ Sixty-two percent of the 135 patients with CPP of unknown origin also reported significantly decreased pain. SHP blockade prior to presacral neurectomy may offer good predictive value. In one report, 10 of 11 patients who underwent successful SHP block experienced greater than 50% pain relief after neurectomy.²⁰

Laparoscopic uterosacral nerve ablation (LUNA) represents another surgical option. This procedure involves the excision of the IHP (a coalescence of sympathetic and parasympathetic nerves at the base of the broad ligament). Although few studies have evaluated this procedure and no randomized trials have been performed, preliminary data are promising. For example, a small study showed that 81% of 21 patients with CPP experienced decreased pain after a uterovaginal ganglion excision.²¹

A final approach is neurosurgical and creates lesions in the central nervous system. Several case reports demonstrate success in treating incapacitating pelvic pain.² However, risks are high, and it is unlikely that large clinical studies will be performed to evaluate its efficacy because surgeons rarely resort to this technique.

Neuromodulation (Sacral Nerve Stimulation)

Sacral stimulation has been used to successfully treat voiding dysfunction for several years. For instance, some patients suffering from interstitial cystitis have benefited from electrical modulation of the sacral nerves and report improvement in pain and urinary urgency. Recently, pain physicians have applied the technique for the treatment of CPP. The transforaminal approach consists of a trial stimulation in which an electrode is placed percutaneously into the S3 or S4 foramen in the area of the nerve roots (Fig. 29–2). If the trial is successful, the patient is taken to the operating room, where the permanent lead is placed and tunneled subcutaneously to an implanted pulse generator. In an observational study of 10 patients undergoing sacral nerve stimulation for intractable pelvic pain, 9 reported a decrease in pain severity for at least 19 months.²² In another study, 11 patients were followed for 36 months after undergoing sacral stimulator placement; 9 experienced extended and significant reduction in their pelvic pain, and 2 failed the therapy soon after implantation.²³ This failure was likely due to a false-positive result during the trial. These preliminary studies suggest that



Figure 29-2. Anteroposterior (AP) fluoroscopic view of sacral stimulation: bilateral leads over the sacral nerves.

sacral stimulation may be helpful in reducing pelvic pain among properly chosen patients who undergo a successful stimulator trial

Technique

Psychiatric comorbidities, substance misuse/abuse, and issues of secondary gain should be assessed prior to implementation. This examination is usually performed by a licensed psychologist. Test stimulation with a temporary lead is performed under fluoroscopic guidance. The lead is placed through the sacral hiatus and advanced in an antegrade fashion toward the sacral foramen or inserted through the low lumbar vertebrae and advanced retrograde toward the sacral foramen. The lead is then positioned with contacts placed inside the S3 or S4 foramen, and test stimulation is performed to ensure coverage over the intended pelvic region. A dressing is placed over the insertion site to anchor the lead as well as to prevent infection. A trial of stimulation is then performed on an outpatient basis for 3 to 5 days. If the patient reports a significant reduction in pain during the trial (typically considered at least a 50% reduction), a permanent stimulator implantation is offered. The implantation is performed in an operating room. During the surgery, the percutaneous leads are anchored to underlying fascia, tunneled underneath the skin, and attached to the pulse generator (battery), which is placed in a subcutaneous pocket. Several locations are suitable for placement of the pulse generator including the upper buttock or inferior to the last rib anteriorly.

Complications

Spinal cord injury or nerve injury, cerebrospinal fluid leak, infection, bleeding, lead migration/malposition, lead fracture, epidural abscess, and generator failure may occur.

Contraindications

Patient refusal, sepsis, coagulopathy, pregnancy, untreated psychiatric comorbidities (anxiety, depression), substance abuse/misuse, inability to cooperate, secondary gain, demand cardiac pacemaker

(need to change to a fixed rate), specific needs for magnetic resonance imaging (MRI; spinal cord stimulation [SCS] equipment is incompatible with MRI).

Neuromodulation (SCS)

SCS has been used for the treatment of lumbosacral radicular pain after spine surgery, intractable cardiac ischemia, peripheral vascular disease, occipital neuralgia, and complex regional pain syndrome. There is emerging evidence that a midline dorsal column pathway exists that may mediate the perception of visceral pelvic pain; therefore, dorsal column stimulation may serve an effective means of treating CPP (Fig. 29-3). For instance, Kapural and colleagues²⁴ reported that six female patients with severe CPP undergoing dual-lead implantation with the lead tip between the levels of T11–12 described significant improvement in pain scores and activities of daily living during an average follow-up of 2.6 years. Although the study was retrospective and small, it provided a framework for other researchers to clarify the efficacy of SCS in visceral pelvic pain with randomized, controlled trials.

Nonsurgical Interventions

Neurolysis

Neurolysis refers to the intentional injury of a nerve or nerves with the intent of reducing pain. Pain physicians may offer nonsurgical neurolytic treatments with chemicals (alcohol or phenol), cryoablation, or thermocoagulation. Many practitioners reserve these treatments for cancer-related pain because of the associated risks such as excessive neurologic injury, damage to nonnervous tissue, and spotty relief owing to tumor or scar tissue. For example, SHP ablation has demonstrated a 69% percent success rate for 6 months in cancer patients with intractable pelvic pain. In patients with



Figure 29-3. AP fluoroscopic view of the thoracolumbar spine with two leads positioned midline from T10–12 in anticipation of spinal cord stimulator implantation.

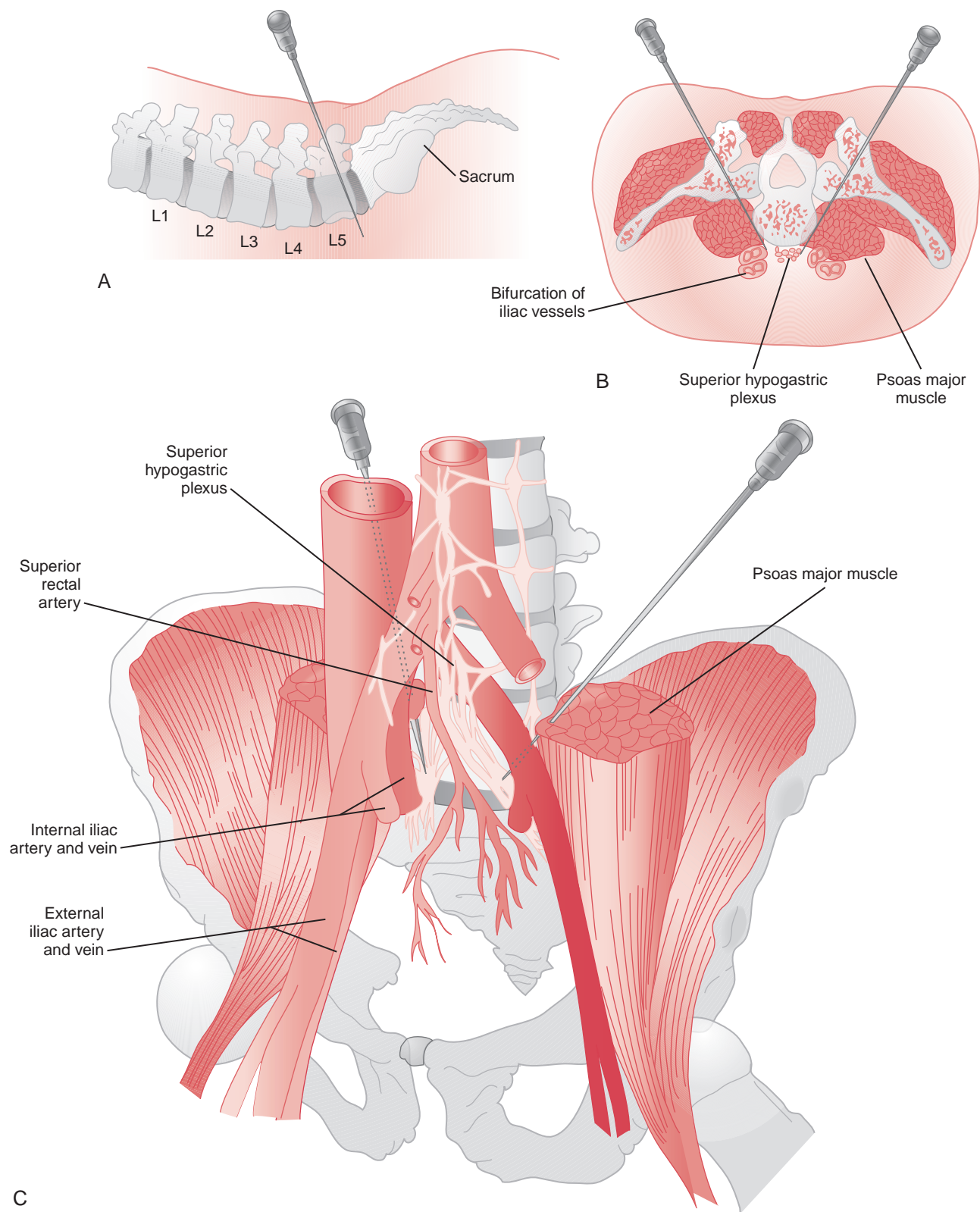


Figure 29-4. Anatomic approach to the superior hypogastric plexus block. **A.** Patient positioned prone with the needle positioned to the infero-lateral portion of the L5 vertebral body and the needle tip directed to the L5-S1 interspace. **B.** Cross-sectional view of the SHP with bilateral needle tips inserted toward the anterolateral aspect of L5, through the psoas major, and medial to the iliac vessels. **C.** Anterior and oblique view of the lumbar spine and pelvis demonstrating bilateral needle approach to the SHP and associated anatomy. (From Cousins MJ, Bridenbaugh PO [eds.] *Neural Blockade*, 3rd ed. Philadelphia, Lippincott-Raven, 1998.)

nonmalignant pain, sequential SHP blockade with local anesthetics may attenuate central sensitization and sympathetically maintained pain, resulting in prolonged relief. Ganglion impar denervation has been performed in cancer patients with persistent perineal or rectal pain, but randomized, controlled trials to support this therapy have not yet been performed. Pain physicians treating CPP due to cancer should consider pretreatment with local anesthetics prior to injection of alcohol for neurolysis owing to significant pain on injection and burning or shooting sensations after injection. Unlike alcohol, phenol exhibits local anesthetic properties that render the injection painless.

SHP Block

Indications

Pelvic pain related to nonmalignant conditions or cancer can be reduced by blocking the SHP. Patients suffering from CPP associated with endometriosis, adhesions, inflammation, interstitial cystitis, irritable bowel syndrome, and chronic pain related to the bladder, prostate, testes, uterus, ovaries, descending/transverse colon, vagina, rectum, postsurgical pelvic pain, or neoplasms of the pelvis should be considered candidates. Afferent fibers innervating the pelvic viscera travel with the sympathetic nerves, trunks, ganglia, and rami; thus, a sympathectomy can produce analgesia from painful pelvic structures. SHP blocks can be used for either diagnostic or therapeutic purposes. For instance, the block may help differentiate between low back pain referred from chronic pelvic disease and low back pain from myofascial, facet, disk, or other primary lumbar pathology.

Anatomy (Fig. 29-4)

The SHP lies in the retroperitoneum, anterior to the sacral promontory, in proximity to the bifurcation of the common iliac vessels, and extends from the lower third of the fifth lumbar vertebral body to the upper third of the first sacral vertebral body (L5–S1 interspace). It exists as a weblike convergence of interconnecting fibers that carry both sympathetic fibers and parasympathetic fibers (S2–4). The ureter is located close to the anterolateral aspect of the L5 vertebral body.

Technique

The patient is positioned prone in a comfortable position with pillows beneath the abdomen to flatten the lumbar lordosis. The low back is prepared and draped in a sterile fashion. The SHP can be accessed through a posterior paravertebral (lateral), transdiskal, transvascular, or transvaginal approach under fluoroscopic or computed tomography (CT) guidance. The more common paravertebral and transdiskal approaches are described. In the lateral approach, the L4–5 interspace is identified by palpating iliac crests and confirmed with fluoroscopy. The skin and subcutaneous tissues are anesthetized with local anesthetic 5 to 7 cm lateral to the midline on each side at the L4–5 level. Two, 7-inch, 22-gauge needles are directed 45° medially and 30° caudally under image guidance until the tips lie anterolateral to the L5–S1 interspace. Biplanar fluoroscopy (anteroposterior [AP] and lateral images) is needed to confirm proper placement. If the transdiskal approach is used (Fig. 29-5), one 7-inch, 22-gauge needle is typically inserted through the L5–S1 disk to approximate the L5–S1 junction anteriorly or slightly anterolaterally. After negative aspiration, 2 to 3 ml of radiographic contrast is injected through each needle to verify proper needle position and spread of material along the anterior surface of the lumbosacral junction and at the midline region under AP view (Figs. 29-6 and 29-7). A total of 6 to 8 ml of 0.25% bupivacaine is then injected through each of two needles or 12 to 15 ml through

one needle in the transdiskal approach. If patients report meaningful relief (at least 50% pain reduction) and chemical neurolysis is desired, 6 to 10 ml of 6% to 10% phenol or 50% to 80% alcohol is injected through each needle. If a lateral approach is preferred, two needles are required to achieve adequate coverage of the plexus with local anesthetic or chemical neurolytic (Figs. 29-8 and 29-9).

Complications

Somatic nerve injury, cerebrospinal fluid leak, infection, vascular puncture of common iliac vessels, disk trauma, diskitis, hypotension, intrathecal/epidural injection, bladder and ureteral trauma may occur.

Contraindications

Patient refusal, sepsis, coagulopathy, pregnancy, untreated psychiatric comorbidities (anxiety, depression), inability to cooperate are contraindications.

Ganglion Impar Block

Indications

Visceral or sympathetically maintained perineal pain from the rectum, anus, distal urethra, vulva, and distal third of the vagina of malignant or nonmalignant origin are indications. Patients may report burning sensations and urgency.

Anatomy

The ganglion impar is a semicircular, solitary, retroperitoneal structure often positioned midline at the level of the sacrococcygeal junction and marks the end of the bilateral sympathetic chains. The impar contains gray rami communicantes that travel to sacral and coccygeal nerves, although the precise neural interconnections are incompletely understood. The impar seems to lack white rami communicantes. Visceral afferents that innervate the perineum, rectum, anus, distal urethra, vulva, and distal third of the vagina travel to the ganglion impar.

Technique

Most often, the patient is positioned prone on the fluoroscopy table. The anococcygeal region is prepared and draped in a sterile fashion. A 22-gauge, 3.5-inch needle is manually bent about 1 inch from its hub to form a 25° to 30° angle. This bend facilitates needle position toward the anterior aspect of the sacrococcygeal concavity. Lateral fluoroscopic imaging is used to identify the coccygeal area, and the needle is introduced under local anesthesia through the anococcygeal ligament or an intercoccygeal space (Fig. 29-10). If the intercoccygeal space is used, the needle tip may require only a slight bend around its tip. The needle tip is then directed anterior to the coccyx but close to the anterior surface of the bone until it reaches the sacrococcygeal junction. The interventionalist should pay close attention to needle depth to avoid rectal trauma because this structure lies close to the sacrum (Fig. 29-11; see also Fig. 29-10). Midline position is confirmed with an AP view (Fig. 29-12). After negative aspiration of blood or stool, 2 to 3 ml of contrast is injected to confirm retroperitoneal location (see Fig. 29-11). Laterally, the contrast should appear smoothly contoured and hug the coccygeal concavity like a teardrop (see Fig. 29-11). For diagnostic or therapeutic purposes, 4 to 6 ml of 0.25% bupivacaine or 1% lidocaine is injected. For neurolytic blockade, 4 to 8 ml of 6% to 10% phenol or 50% to 80% alcohol is injected.

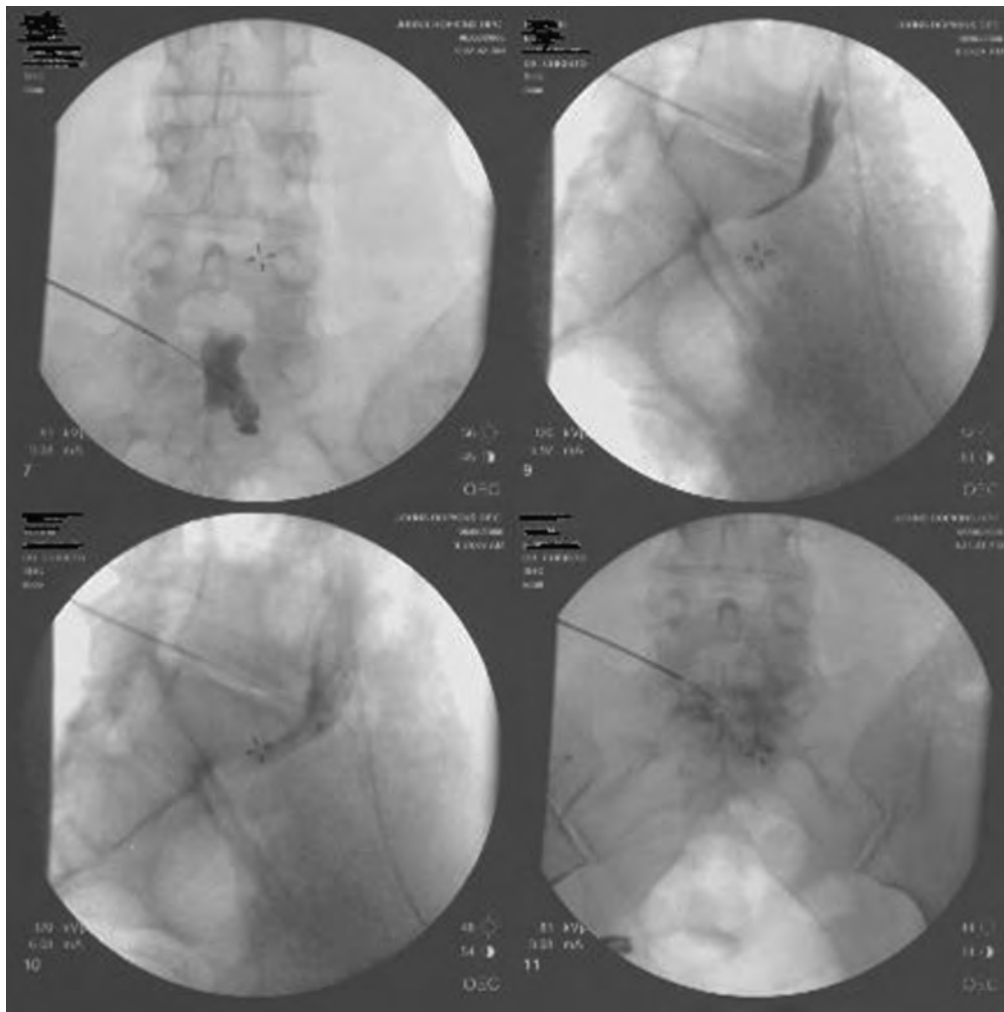


Figure 29-5. Transdiskal, single-needle approach to the superior hypogastric plexus under fluoroscopy. Images show AP and lateral views depicting the proper spread of contrast.



Figure 29-6. Two-needle approach to the superior hypogastric plexus and the proper spread of contrast material in the AP fluoroscopic view.



Figure 29-7. Lateral fluoroscopic view of two needles positioned at the L5-S1 junction with proper spread of contrast along the region of the superior hypogastric plexus.



Figure 29-8. Bilateral needle approach to the superior hypogastric plexus under AP fluoroscopic imaging.

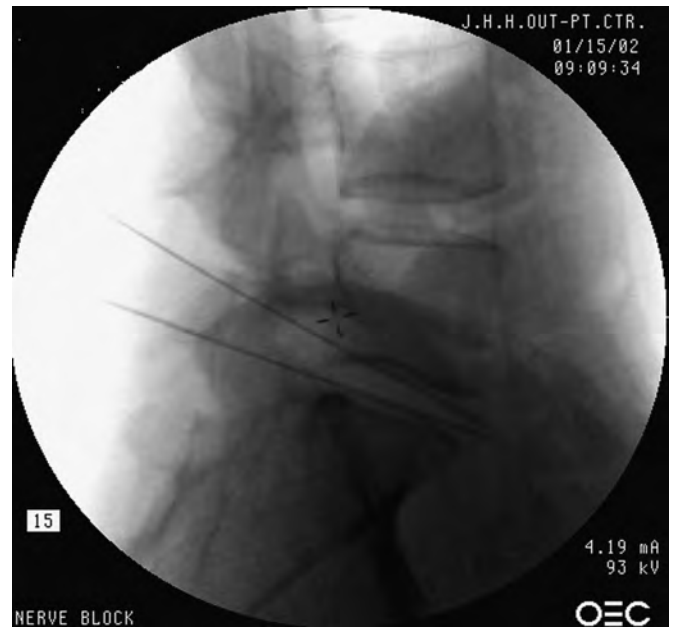


Figure 29-9. Two needles inserted bilaterally and approaching the region of the superior hypogastric plexus at the L5–S1 junction under the lateral fluoroscopic view.

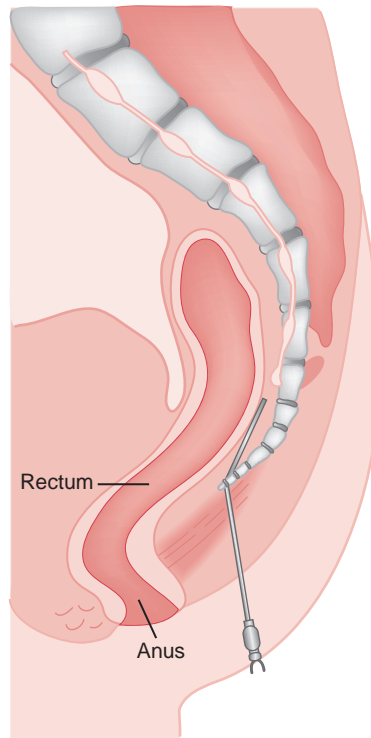


Figure 29-10. Schematic of the ganglion impar and relevant anatomy. (From Benzon HT, Raja SN, Borsook D, et al [eds]. *Essentials of Pain Medicine and Regional Anesthesia*. Philadelphia: Elsevier, 1999; p330.)



Figure 29-11. Lateral fluoroscopic image of a needle through the intercoccygeal joint and retroperitoneal contrast spread resembling a teardrop. Note the presence of bowel gas anterior to the contrast medium.

Complications

Sacral nerve root injury (neurolysis), rectal perforation, periosteal injection, and epidural spread may occur.

Contraindications

Patient refusal, sepsis, coagulopathy, pregnancy, untreated psychiatric comorbidities (anxiety, depression), inability to cooperate, local infection are contraindications.



Figure 29-12. AP fluoroscopic view of contrast spread at the midline and moving superiorly.

Levator Ani Syndrome

Patients exhibit dull, aching, or pressure-like sensations in the rectum that may last for hours. Diagnosis is made if patients report chronic (at least 3 mo) or recurrent bouts of rectal pain that last 20 minutes or longer without identifiable causes such as coccydynia, hemorrhoids, anal fissures, sphincter abscess, cryptitis, ischemia, or inflammatory bowel disease. Methods of treatment include sitz baths, digital massage three to four times a week, and muscle relaxants (diazepam or methocarbamol).

Proctalgia Fugax

Symptoms include episodes of severe, sudden, stabbing, cramping, or aching rectal pain that lasts from seconds to minutes. The pain usually lasts less than 5 minutes and rarely as long as 30 minutes. Women tend to be more affected than men. Diagnosis requires the absence of anorectal conditions that could produce rectal pain. Treatments that have been utilized include caudal epidural blockade, clonidine, nitrates, diltiazem, and inhaled salbutamol.

Pudendal Nerve Entrapment (Pudendal Neuralgia)

The pudendal nerve derives from the sacral plexus (S2–4) and enters the gluteal region via the greater sciatic foramen. The nerve travels through the pelvis around the ischial spine, between the sacrospinous and the sacrotuberous ligaments. Finally, it divides into three branches: anal/rectal, perineal, and clitoral/penile.

Pudendal neuralgia is often attributable to mechanical or inflammatory damage to the nerve caused by pressure or trauma. Pudendal nerve entrapment is sometimes referred to as *cyclist's syndrome*. Symptoms may include a stabbing, burning, or pinpricking sensation in the penis, scrotum, labia, perineum, or anorectal region. Sometimes, the pain may refer to the groin, medial thigh, buttock, and abdomen. Patients report that sitting and other flexion activities of the hip (sitting, squatting, cycling, exercising) exacerbate the pain whereas standing or lying down relieves the discomfort. The distribution may be ipsilateral or even bilateral, and patients may demonstrate allodynia and hyperalgesia in the affected region. There may be associated urinary, anal, or sexual dysfunction.

Treatments consist of using a doughnut-shaped pillow to reduce the pressure on the pudendal nerve, antiepileptic or TCA medications, image and nerve stimulator-guided pudendal nerve blocks positioned at the ischial spine or Alcock's canal (pudendal canal) with local anesthetic/steroid, and surgical decompression of the pudendal nerve.

Coccygeal, Ilioinguinal, Genitofemoral Blocks

These blocks may be performed owing to pain from coccygeal trauma or abdominal surgery.

CONCLUSIONS

CPP remains obscure in its diagnosis and treatment modalities. Because the pelvis is complex neuroanatomically and neurophysiologically, pelvic pain may exhibit mixed characteristics of somatic, autonomic, visceral, and neuropathic origin. Underlying pathogenic pain mechanisms in CPP require a variety of treatment strategies including medications, physical restoration, behavioral medicine techniques, surgery, neuromodulation, and SHP and ganglion impar blocks/neurolysis. A thorough systematic evaluation with proper screening for coexisting diseases is critical to

formulating a rational treatment plan. Furthermore, a multidisciplinary approach to patients with CPP aids in coordinating the assessments of primary care physicians and specialists in pain medicine, urology, gynecology, gastroenterology, physiotherapy, and psychology. In fact, a randomized clinical trial by Peters and coworkers²⁵ compared the effectiveness of a focused organic approach to CPP and a multidisciplinary approach consisting of surgical, psychological, social, and dietary interventions. Patients in the multidisciplinary treatment group reported greater improvement in self-reported pain scores.

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Chapter 30

FEMALE PERINEAL/PELVIC PAIN: THE REHABILITATION APPROACH

Colleen M. Fitzgerald and Christina K. Hynes

INTRODUCTION

Physiatrists since the early days of physical medicine and rehabilitation have often been the physicians who take over where others have left off. They are doctors who need to formulate treatment strategies for those patients whose diagnoses leave others confused and frustrated. Certainly, persistent pelvic pain has fallen into this category. Often considered the “black box” of medicine, it is this author's belief that understanding the pelvic anatomy and

physiology is what clears the path for accurate diagnosis and treatment of pelvic pain. In the past, visceral etiologies and psychogenic causes topped the list. Current thinking incorporates musculoskeletal diagnoses predominated by dysfunction of the pelvic floor musculature. Whether the initial insult is visceral or not, the somatic response can become the ultimate culprit, particularly in the persistence of pelvic pain. Perhaps if acute pelvic pain musculoskeletal diagnoses were made more definitively, chronic pelvic pain (CPP) would not be as rampant. Some research supports that pregnancy- and postpartum-related pelvic pain can be the initial insult for those who develop chronic issues. This chapter explores the role of the musculoskeletal system in the diagnosis of pelvic pain, reviews pelvic musculoskeletal anatomy, identifies physical examination techniques in making the pelvic pain diagnosis, and proposes effective rehabilitation treatment options in the management of pelvic pain.

PELVIC ANATOMY AND FUNCTION

Muscle/Fascia

The pelvic floor is composed of layers of muscles connecting the anterior and posterior pelvic ring and surrounding the urethra,